PROCEEDINGS OF THE BRITISH CARDIAC SOCIETY

An AUTUMN MEETING of the British Cardiac Society was held at the Royal Society of Medicine, 1, Wimpole Street, London, on Friday October 30, 1953. The Chairman, SIR JOHN PARKINSON, took the Chair at 9.30 a.m. ; 124 members and 32 visitors were present.

PRIVATE BUSINESS

The minutes of the last Annual General Meeting, having been published in the Journal (**15**, 461, 1953) were taken as read and confirmed.

It was agreed that the Annual General Meeting would be held on Friday, April 9, 1954, at Cambridge. Members would be accommodated in Downing College by kind permission of Sir Lionel Whitby and the Fellows. Papers would be presented in the Large Anatomy Lecture Theatre by kind permission of Professor J. D. Boyd. The Annual Dinner would also be held in Downing College on April 9.

Further discussion took place concerning the Second World Congress of Cardiology to be held in Washington in September, 1954. The Secretary stated that he had received provisional notification that 36 members wished to attend the Congress.

SHORT COMMUNICATIONS

PATENT DUCTUS ARTERIOSUS : THE EFFECTS OF DUCTAL LIGATION ON THE SYSTEMIC BLOOD PRESSURE

By D. LEWES. Observations on the blood pressure were made in a series of 26 cases of patent ductus arteriosus immediately before and after surgical ligation of the ductus, during the first fortnight after operation, and at intervals for the next four years. Anæsthesia and surgical intervention before ligation caused a significant increase in the diastolic pressure but little change in the systolic pressure. Closure of the ductus produced an immediate further increase in diastolic pressure.

Within 24 hours of operation the diastolic and systolic pressures increased significantly, especially the systolic pressure. Blood pressure readings fell slowly for the next 10 days after operation. Two weeks after, the mean systolic pressure had fallen to pre-operative levels; the mean diastolic pressure had fallen by approximately 30 mm. but remained 20 mm. above the level recorded before operation.

The subsequent blood pressure records made during the next four years indicated that the diastolic pressure, obtained two weeks after operation, was maintained but that the systolic pressure continued to fall.

The results of this investigation suggest that the long-term effects of ductal ligation on blood pressure are: (*a*) permanent abolition of a mild but significant pre-operative systolic hypertension, (*b*) stabilization of the diastolic pressure within two weeks of operation, and (*c*) permanent elevation of the diastolic pressure above normal.

The appearance of a continuous murmur eleven days after operation in one case was accompanied by a profound fall in the diastolic pressure to pre-operative levels and by collateral evidence of ductal recanalization. Despite the persistence of the murmur, the diastolic pressure observed during a three-year period, steadily increased. From the evidence obtained in this and in comparable cases recorded by Gilchrist (1945) it is suggested that measurement of the diastolic blood pressure following the appearance of a continuous murmur after ductal ligation may give more accurate information as to the degree of patency of the ductus than does the character of the murmur accompanying it.

In the present series of 26 cases a significant positive correlation between the resting pulse pressure and the ductal diameter measured at operation has been found.

Patent Ductus Arteriosus with Systolic Flow inhibited by Pulmonary Hypertension

By Peter Harris (*introduced by Terence East*). A lady of 43 had been noted to have an abnormal heart at 8 but there had been no disability until 42, when she had several attacks of paroxysmal fibrillation and increasing dyspnœa with, finally, heart failure. Clinically she had no cyanosis, a collapsing pulse, a right ventricular thrust, and a loud diastolic murmur in the pulmonary area. Systole was silent. Radiology revealed the pulmonary trunk and main branches dilated and pulsatile, with enlargement of both ventricles.

Cardiac catheterization showed a pressure of 140/10 mm. Hg in the pulmonary artery where the blood was excessively oxygenated. Femoral artery pressure was 140/20, and it was thought that blood flow through a patent ductus was occurring only during diastole, since there was no pressure gradient along it during systole. There was no evidence of reversed flow. The pulmonary hypertension was not simply due to increased flow for the pulmonary arteriolar resistance was raised. The low diastolic pressure was due to pulmonary incompetence. Angiocardiography confirmed the great dilatation of the pulmonary trunk and main branches, the peripheral branches being narrowed. Subsequent enquiry revealed that at 9 she had a pulmonary systolic murmur only, while at 25 there was a typical continuous murmur.

A patent ductus was found post mortem. The natural history, physiology, and morbid anatomoy are discussed and correlated.

Sodium Chloride and Cardiac Muscle

By R. J. S. McDowall and A. F. Zayat (*introduced*). It has long been recognized that low sodium chloride is of benefit in cardiac œdema because it causes less water to be retained in the body. The introduction of the rat ventricle preparation (McDowall and Zayat, 1952, *J. Physiol.*, **117**, 75P) indicates clearly that it may be of great benefit to cardiac muscle, especially when hypoxic.

If the preparation is rendered anoxic for a few minutes the cardiac muscle is markedly disabled. Recovery, which otherwise is very slow, may be brought about immediately by reducing the sodium chloride in the external medium and so facilitating sodium extrusion. It is known that all muscle contains less sodium than the surrounding medium in virtue of a sodium extruding mechanism, but retains it when anoxic. We have also evidence that the power of the heart to contract very rapidly is dependent on the amount of sodium in the external medium and it is suggested that cardiac muscle, when it contracts takes up sodium, in the same way as nerve when it conducts.

Systolic Murmurs in Mitral Stenosis

By J. P. D. Mounsey. Systolic murmurs were examined by auscultation and on the phonocardiogram in 50 patients with mitral stenosis and in these the degree of stenosis and the degree of associated incompetence, if any, was estimated at mitral valvotomy. Systolic murmurs were met in approximately two-thirds of the patients and were absent in the remaining one-third.

Pan-systolic murmurs, best heard at the apex, were in every case associated with mitral incompetence. Although, in general, louder murmurs were associated with a greater degree of incompetence, this was not an invariable finding. Of soft systolic murmurs, confined to early and mid systole, best heard at the apex, one-third were associated with palpable mitral incompetence. In the remaining two-thirds no regurgitation was felt.

Where an apical systolic murmur was absent on auscultation and on the phonocardiogram, no incompetence was palpable at operation.

Transient, loud, pan-systolic murmurs, heard well at the apex but loudest at the left border of the sternum at the level of the fourth inter-costal space, were met during episodes of congestive cardiac failure. These lessened or disappeared after recovery from failure and were not necessarily associated with incompetence of the mitral valve. Likewise, systolic murmurs, best heard elsewhere than at the apex, were not associated with mitral incompetence.

It is suggested that in mitral stenosis, a pan-systolic murmur, best heard at the apex, in the absence of congestive cardiac failure, is strong evidence of the presence of associated mitral incompetence. The loudness of the murmur, on the other hand, is a less reliable guide to the degree of incompetence, in estimating which the whole clinical picture must be taken into account.

Pulmonary Distensibility and Respiratory Work in Mitral Stenosis

By Graham Hayward and J. M. S. Knott (*introduced*). The pulmonary distensibility and respiratory work have been measured in patients with mitral stenosis before and after valvotomy, at rest and during exercise, by means of simultaneous recordings of intra-œsophageal pressure and respiratory air flow. The

rigidity of the lungs is usually increased at rest and increases further with exercise, but there is no correlation between the extent of these changes and the degree of dyspnœa. Respiratory work shows similar changes.

After valvotomy, the rigidity of the lungs is decreased but the respiratory work on exercise sufficient to cause dyspnœa is the same as before operation. In one patient who seemed considerably improved by operation although no valvotomy was technically possible, the lungs were more rigid after the operation than before. The results of mitral valvotomy suggest that the improved ability to carry out muscular and respiratory work may play as important a part as the decrease in pulmonary congestion in causing symptomatic improvement after operation.

Criteria for the Diagnosis of Lutembacher's Syndrome

By Lawson McDonald. The observations in this communication were made in association with Dr. Lewis Dexter, Florence Haynes, Murray Rabinowitz, and George A. Saxton, Jr. The diagnosis of mitral stenosis in the presence of atrial septal defect is important when either lesion may be corrected by surgery.

Clinical, electrocardiographic, and radiological findings are given in a case of atrial septal defect in whom mitral stenosis, as a complicating lesion, could not be excluded before operation. Venous catheterization demonstrated the presence of an atrial septal defect, and the left atrial mean pressure was raised to 17 mm. Hg. As this could have been due either to mitral stenosis or to left ventricular failure, repeated but unsuccessful attempts were made to pass the catheter through the mitral valve into the left ventricle in order to obtain pressure reading there. At operation a large atrial septal defect was found; there was no mitral stenosis.

In such cases where mitral stenosis is suspected but not proved (as by the demonstration of mitral calcification), left ventricular pressure records may be of diagnostic value. These will show whether an elevated left auricular pressure is due to obstruction at the mitral valve or to left ventricular failure. If the catheter cannot be made to enter the left ventricle, after being passed into the left auricle through an atrial septal defect, arterial catheterization may be indicated. This has not yet been successfully performed in these cases, although it has been in others.

The criteria for the diagnosis of mitral stenosis occurring with atrial septal defect are reviewed, with special reference to the above and to two other similar cases. Of these, both underwent cardiotomy and one was also examined post mortem.

Cardiac Output during Exercise by the Fick Method

(Illustrated by film)

By K. W. Donald, O. L. Wade, J. M. Bishop, and G. Cumming *(introduced)*. To use the Fick method to estimate cardiac output during such a changing state as the transition from rest to exercise requires very frequent samples of arterial and mixed venous bloods and expired air. Furthermore, in a changing state the procedure is useless if each set of samples is not simultaneous. The film shows how this can be done, and demonstrates a spectrophotometric method of measuring blood oxygen content and capacity which allows the analysis of 50 specimens in duplicate in three hours.

Non-specific Factors in the Prothrombin (Quick's) Test

By R. Hudson. The one-stage prothrombin-test is at present the only practical means of controlling anticoagulant therapy with the modern coumarin drugs. The test is relatively insensitive to changes in prothrombin level but fortunately it is sensitive to changes in factor VII—and it is this factor that is lowered by the drugs.

Plasma is mixed with brain suspension at 37° C. for a fixed time and then, after the addition of calcium chloride solution (also at 37° C.), the time taken for clotting to occur is the prothrombin time. The whole technique of the test must be standardized. The prothrombin time of a patient's plasma is interpolated on a normal plasma-saline dilution curve and the result expressed empirically as a percentage of prothrombin activity on a saline-dilution curve.

Though empirical, this technique provides a reliable guide to therapy and this is borne out by results —nearly 170 patients have received courses of therapy with only one hæmorrhage—and this accident was anticipated by the test.

Apart from heparin administration, and *in vivo* deficiencies of pro-factor VII, prothrombin, factor V, and fibrinogen, abnormal prothrombin times can arise *in vitro* from several causes which may be set out as follows.

Factors that have little effect on prothrombin time

1. Moderate hæmolysis of blood sample.
2. Minor degrees of clotting in blood sample.
3. Excessive platelets in the plasma used for the test.

Factors that may cause major error

1. Incorrect ratio of blood to potassium oxalate in sampling. Too little blood results in prolonged prothrombin time.
2. Age of sample. Whole blood retains its prothrombin time unchanged for at least three hours when kept at room temperature or in refrigerator. Separated plasma shows considerable lengthening of prothrombin time if kept at room temperature—but less so in refrigerator—for three hours.
3. Incorrect water bath temperature. Prothrombin times carried out below 37° C. show prolongation. This effect is noticeable at 30° C. and gross at room temperature.
4. Addition of calcium solution too soon produces shortened prothrombin times which vary with the incubation time and the thickness of tube used. Uniform tubes and an incubation time of one minute before adding calcium solution yields consistent results.
5. Brain extract unstable or improperly standardized. The dried powder stored in vacuum ampoules in refrigerator is absolutely stable for years. When made up for use the suspension must be kept at refrigerator temperature always, and must be used at its optimum dilution.

Auricular Waves on Arterial Pressure Records in Patients with Sinus Rhythm, Heart Block, and Auricular Flutter

By Sheila Howarth. To be published in full in *Brit. Heart J.*

Pulmonary Venous Drainage into the Right Heart via a Persistent Left Superior Vena Cava

By James W. Brown and William Whitaker. Drainage of some or all of the pulmonary veins into the right side of the heart is not uncommon and Muir (*Thorax*, **8**, 65, 1953) collected 151 reports of such cases. However, it is only since the advent of cardiac catheterization and angiocardiography as diagnostic procedures that it has been possible to establish the diagnosis of this lesion in life. Recent reports indicate that it should now be possible to recognize patients with pulmonary veins draining via a persistent left superior vena cava and the left innominate vein into the right heart, by the ordinary clinical and radiological methods. (Friedlich, A. *et al*, *Bull Johns Hopkins Hosp.* **86**, 20, 1950; Snellen, H. A., and Albers, F. H., *Circulation*, **6**, 801, 1952; and Gardner, F., and Oram, S., *Brit. Heart J.*, **15**, 305, 1953.)

Six patients in whom a diagnosis of such anomalous pulmonary venous drainage was made on clinical and radiological grounds are described and the results of cardiac catheterization and angiocardiographic studies in five of these patients are discussed. The possibility of surgical correction is considered.

The Vascular Pattern in Chronic Generalized Myocardial Ischæmia

By W. F. M. Fulton (*introduced by Rae Gilchrist*). From a detailed pathological investigation of coronary artery disease with clinical correlation, four cases have been selected for presentation. These four have in common a long history of progressive, unremittent angina. In each there was narrowing of both coronary arteries at, or close to, their ostia. In no case was there evidence of recent occlusion of any vessel.

Stereo-radiography following the injection of a radio-opaque medium revealed unusually advanced vascular changes in the coronary circulation, which presented a pattern that was characteristic. Anastomoses were widespread, with special emphasis on the deeper layers of the left ventricle, where a network of grossly dilated, intercommunicating channels had opened up. These hearts were apparently protected from localized infarction. Despite severe angina, myocardial infarction either did not occur at all, or else occurred in zonal, as opposed to regional, distribution. Multiple focal areas of necrosis formed in aggregate a ring of infarction encircling the inner zone of the left ventricle, as seen in cross-section.

It is suggested that the more pronounced ischæmic change found in the deeper layers of the left ventricle is lagely conditioned by their distance from supply.

The Ballistocardiogram in Coronary Disease

By E. G. Wade and R. M. Fulton (*introduced*). The ballistocardiogram was recorded in 112 patients complaining of chest pain. Patients were grouped clinically into those (*a*) with typical cardiac pain, (*b*) with non-cardiac pain, and (*c*) with pain of doubtful origin. The ballistocardiographic and electrocardiographic findings in these groups are described. It is concluded that the ballistocardiogram is of diagnostic value in cases of chest pain of doubtful significance and of prognostic value in cases of established coronary artery disease.

The Effect of Œstrogens on the Plasma Lipids in Coronary Disease

By M. F. Oliver and G. S. Boyd (*introduced by Rae Gilchrist*). The effect of small doses of œstrogens on the plasma lipids has been studied in 20 hypercholesterolæmic men with cardiographic proof of coronary artery disease. The plasma lipids of the subjects were studied for a control period of 16 weeks, the majority of the subjects then received from 0·2 mg. to 0·6 mg. of ethinyl œstradiol each day for 11 weeks, and identical inert tablets for a further 8 weeks. The mean plasma total cholesterol fell by 25 per cent of the control value during the period of œstrogen administration, and returned approximately to the control value during the period of inert tablet administration. The change occurred almost entirely in the ester fraction. The plasma phospholipids remained more or less constant, and thus the abnormally high mean *plasma total-cholesterol phospholipid* ratio of this group was depressed into the range of normality during the period of œstrogen administration. Plasma lipoprotein patterns have been studied in selected subjects. All experienced gynæcomastia and other side effects after two or three weeks therapy. There was no change in the functional capacity of the subjects during or after œstrogen therapy.

The therapeutic application of this beneficial effect of œstrogens on the abnormal plasma lipid pattern in established coronary artery disease is discussed.

After the meeting of the Society a dinner was held at Simpsons.

As 1953 was the centenary of the birth of Sir James Mackenzie, the President, Sir John Parkinson, was asked to recall some of his memories of Mackenzie; and the account that follows is based on what he said.